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           739 BYSTANDER
        287859 ANTIGEN?
L1          13 BYSTANDER ANTIGEN?
                 (BYSTANDER(W)ANTIGEN?)

=> s l1 and review
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L2           0 L1 AND REVIEW

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=> s l1 and bystander/ti
235 BYSTANDER/TI
L3 7 L1 AND BYSTANDER/TI

=> d bib ab 1-

L3 ANSWER 1 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 96:47869 BIOSIS
DN 98620004
TI **Bystander antigen** presenting cells repair defective antigen presentation by pre-B acute lymphoblastic leukemia (B-All) cells.
AU Cardoso A A; Seamon M J; Schultze J L; Boussiotis V A; Freeman G J; Gribben J G; Nadler L M
CS Dana-Farber Cancer Inst., Boston, MA, USA
SO 37th Annual Meeting of the American Society of Hematology, Seattle, Washington, USA, December 1-5, 1995. Blood 86 (10 SUPPL. 1). 1995. 46A. ISSN: 0006-4971
DT Conference
LA English

L3 ANSWER 2 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 94:226345 BIOSIS
DN 97239345
TI Activation of CD4+ T cells by delivery of the B7 costimulatory signal on **bystander antigen**-presenting cells (trans-costimulation).
AU Ding L; Shevach E M
CS LI, NIAID, National Inst. Health, Bethesda, MD 20892, USA
SO European Journal of Immunology 24 (4). 1994. 859-866. ISSN: 0014-2980
LA English
AB Increasing evidence in both murine and human systems suggests that the interaction of the T cell surface antigens CD28/CTLA4 with their ligand B7 on the antigen-presenting cells (APC) is the critical costimulatory pathway involved in the induction of maximal T cell activation and the prevention of induction of anergy. It has also been demonstrated that efficient induction of clonal expansion of normal CD4+ T cells requires the delivery of the T cell receptor (TCR) ligand and costimulation by the same APC. We demonstrate here that normal murine CD4+ T cells can be efficiently activated by soluble anti-CD3 cross-linked by fixed macrophages and by a costimulatory signal delivered by a bystander APC, B7-transfected L cells. The major factor which determined the ability of an APC to provide costimulation in "trans" was the level of cell surface B7 expression. The requirement for B7 costimulation appears to be at initial stage of TCR engagement since optimal T cell activation was only observed when TCR triggering and B7 costimulatory activity were delivered at same time by different APC. Induction of maximal proliferation of both naive CD45RB-hi and memory CD45RB-lo CD4+ T cells was B7 dependent and both populations of cells responded equally well to the B7 costimulation delivered in "trans". Furthermore. trans-costimulation provided by B7 transfected L cells efficiently prevented the induction of anergy in normal murine CD4+ T cells induced by anti-CD3 cross-linked by fixed-resting macrophages. Addition of exogenous interleukin-2 (IL-2) and IL-7 to the primary

culture in the absence of B7-transfected L cells or addition of IL-2 to the culture containing the B7 transfectant and CTLA41g completely prevented the induction of hyporesponsiveness. These findings raise the possibility that in certain pathological states, CD4+ T cells in vivo may be activated by costimulation delivered by bystander APC.

L3 ANSWER 3 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 90:105457 BIOSIS
DN BA89:54948
TI DIVALENT HAPTEN-INDUCED INTESTINAL ANAPHYLAXIS IN THE MOUSE UPTAKE AND CHARACTERIZATION OF A BYSTANDER PROTEIN.
AU KLEINMAN R E; HARMATZ P R; HATZ R A; BROWN M; ARINIELLO P D; WALKER W A; BLOCH K J
CS PEDIATRIC GASTROENTEROLOGY AND NUTRITION UNIT, BARTLETT EXTENSION 4TH FLOOR, BOSTON, MASS. 02114.
SO IMMUNOLOGY 68 (4). 1989. 464-468. CODEN: IMMUAM ISSN: 0019-2805
LA English
AB We examined the mucosal barrier function during anaphylaxis induced by the hapten N,N'-di-2,4,dinitrophenyl-lysine (di-DNP-lysine) in BDF1 female mice immunized with dinitrophenylated Ascaris-suum extract. Immunized mice were gavaged with 10 mg or 50 mg of ovalbumin (OVA) with or without N,N'-di-2,4-DNP-lysine (di-DNP-lysine). Animals that received di-DNP-lysine underwent anaphylaxis and were observed to have significantly greater serum concentrations of immunoreactive OVA (iOVA) than control mice. The severity of anaphylaxis, which varied with the dose of di-DNP-lysine administered, influenced the uptake of OVA; greater amounts of iOVA were detected in serum of mice undergoing more severe anaphylaxis. On gel permeation of serum from both groups of mice, immunoreactive OVA was found to have a molecular size similar to native OVA. Di-DNP-lysine is a synthetic hapten that reliably induced anaphylaxis in sensitized animals challenged by gavage. Anaphylaxis resulted in the uptake into the circulation of greater quantities of an unrelated protein antigen present in the intestinal lumen. The protein antigen that was taken up into the circulation appeared to be intact and thus may have an influence on the development of the immune response, or lack thereof, to this **bystander antigen**.

L3 ANSWER 4 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 88:408614 BIOSIS
DN BR35:71589
TI PRESENTATION OF ENDOGENOUSLY-SYNTHESIZED VIRAL PROTEINS BY TRANSFECTED OR **BYSTANDER ANTIGEN**-PRESENTING CELLS APC TO MURINE T-HELPER CELLS IN-VITRO.
AU HACKETT C J; YEWDELL J W; EISENLOHR L C; EAGER K; GERHARD W; RICCIARDI R P
CS WISTAR INST., PHILADELPHIA, PA. 19104.
SO SYMPOSIUM ON CELL BIOLOGY OF VIRUS ENTRY, REPLICATION AND PATHOGENESIS HELD AT THE 17TH ANNUAL UCLA (UNIVERSITY OF CALIFORNIA-LOS ANGELES) MEETING ON MOLECULAR AND CELLULAR BIOLOGY, TAOS, NEW MEXICO, USA, FEBRUARY 28-MARCH 5, 1988. J CELL BIOCHEM SUPPL 0 (12 PART C). 1988. 51. CODEN: JCBSD7
DT Conference
LA English

L3 ANSWER 5 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 86:275899 BIOSIS

DN BA82:19762
TI INDUCTION AND PERSISTENCE OF SUPPRESSION OF CONTACT HYPERSENSITIVITY AGAINST **BYSTAN**DER HAPTENS AND ALLOANTIGENS IN RATS.
AU PROP J; HUTCHINSON I V; MORRIS P J
CS NUFFIELD DEP. OF SURGERY, UNIV. OF OXFORD, JOHN RADCLIFFE HOSP., OXFORD OX3 9DU, ENGLAND.
SO CELL IMMUNOL 99 (1). 1986. 85-94. CODEN: CLIMB8 ISSN: 0008-8749
LA English
AB The shift of suppression from a tolerizing hapten to a so-called **bystander antig**en was investigated in this study using contact hypersensitivity to trinitrochlorobenzene (TNCB) and dinitrofluorobenzene (DNFB) and delayed type hypersensitivity (DTH) to alloantigens in the rat as experimental models. Primary suppression of contact hypersensitivity was induced by intravenous injection of the water-soluble forms of TNCB and DNFB. A shift of the suppression to the bystander hapten was found if the tolerizing and bystander hapten were mixed and applied to the same area of skin during the sensitization procedure, but not if they were applied to separate areas of skin. With alloantigens, bystander suppression developed only when the sensitizing allogeneic cells were mixed with hapten-modified syngeneic cells. It was not induced by hapten-modified allogeneic cells. Once induced, such bystander suppression of the response to haptens persisted independently of the primarily tolerizing hapten, and it could be adoptively transferred with spleen cells. These results favour the concept that the bystander suppression is mediated by the non-specific action of suppressor cells generated specifically during the mixed sensitization rather than by an antigen bridge.

L3 ANSWER 6 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 84:202532 BIOSIS
DN BA77:35516
TI 2 CLASSES OF **BYSTANDER** B CELL RESPONSE ACTIVATION REQUIREMENTS REFLECT THOSE OF B CELLS IN GENERAL.
AU RATCLIFFE M J H; JULIUS M H
CS BASEL INST. IMMUNOLOGY, GRENZACHERSTR. 487, CH-4005 BASEL, SWITZ.
SO J IMMUNOL 131 (2). 1983. 581-586. CODEN: JOIMA3 ISSN: 0022-1767
LA English
AB Bystander anti-sheep red blood cell (SRBC) antibody responses were induced in cultures containing unprimed B cels, SRBC, particulate or soluble eliciting antigen and helper T (Th) cells primed to the eliciting antigen. The use of long-term cultured Th cells, depleted of alloreactive cells, allowed direct assessment of the requirements for major histocompatibility complex- (MHC) restricted interactions in the cellular events leading to bystander B cell activation. H-2 restricted Th cell activation limited all bystander B cell responses; the requirement for H-2 restricted Th-B cell interactions varied with the state of activation of the responding B cell population before assay. Two classes of bystander B cell response were defined. Activation of small (i.e., resting) SRBC-specific bystander B cells, purified by density gradient centrifugation, was completely restricted by B cell MHC. The requirement for high concentrations of eliciting antigen suggests that nonspecific binding of soluble or particulate antigen to the responding B cell surface plays a mandatory role leading to the activation of resting bystander B cells. The concomitant requirement for a restricted Th-B cell interaction suggests that this role is one of focusing

antigen-specific, H-2 restricted T cell help to the resting B cell. Bystander responses, unrestricted by B cell MHC, were generated exclusively from large (i.e., blasted) B cells limited only by Th cell activation. This class of SRBC-specific bystander response correlated with an SRBC-dependent increase in the number of cells secreting antibodies that recognized degraded mouse RBC. Apparently, the H-2 unrestricted anti-SRBC response is derived from the amplification of B cells activated in vivo to degraded homologous RBC. Both classes of anti-SRBC response were enhanced by the presence of the **bystander antigen**. This phenomenon is discussed in terms of a mitotic advantage provided by the interaction of antigen with surface Ig on specific bystander B cells.

L3 ANSWER 7 OF 7 BIOSIS COPYRIGHT 1996 BIOSIS
AN 81:250571 BIOSIS
DN BA72:35555
TI EFFECT OF LOCALLY INDUCED INTESTINAL ANAPHYLAXIS ON THE UPTAKE OF A **BYSTANDER ANTIGEN**.
AU BLOCH K J; WALKER W A
CS CLIN. IMMUNOL., ALLERGY UNITS, MASS. GEN. HOSP., BOSTON, MA 02114.
SO J ALLERGY CLIN IMMUNOL 67 (4). 1981. 312-316. CODEN: JACIBY ISSN: 0091-6749
LA English
AB Adult Sprague-Dawley rats weighing 250 g were immunized with 100 .mu.g of egg albumin (EA) and alum. Intestinal anaphylaxis was induced by intraduodenal or intragastric challenge with EA 14 days later. To test for the changes in vascular and mucosal permeability that accompany anaphylaxis, the rats were injected i.v. with 125I-rat serum albumin (RSA) at the time of challenge; The amount of radioactivity retained in the wall of gut segments and the trichloroacetic acid (TCA)-precipitable radioactivity present in the secretions obtained from these segments was determined. Enhanced retention of 125I-RSA was found in the wall of the duodenum and 1st and 2nd halves of the small intestine, and increased amounts of TCA-precipitable radioactivity was found in gut segments obtained from antigen-challenged compared with buffer-challenged rats. In other EA-immunized rats, bovine serum albumin (BSA) was administered by gavage 1 h before challenge with EA. Increased amounts of immunoreactive BSA were detected by radioimmunoassay in the serum of rats subjected to local intestinal anaphylaxis. Possible consequences of the enhanced systemic uptake of protein present during local intestinal anaphylaxis were considered. Such proteins might induce an IgE antibody response, broadening the anaphylactic sensitivity of the animal to additional antigens.

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L1 0 S BYSTANDER ANTIGEN
L2 272 S BYSTANDER
L3 20 S L2 AND ANTIGEN
L4 13 S L3 AND AUTOIMMUNE

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US PAT NO: 5,580,756 [IMAGE AVAILABLE] L4: 1 of 13
DATE ISSUED: Dec. 3, 1996
TITLE: B7IG fusion protein
INVENTOR: Peter S. Linsley, Seattle, WA
Jeffrey A. Ledbetter, Seattle, WA
Nitin K. Damle, Renton, WA
William Brady, Bothell, WA
ASSIGNEE: Bristol-Myers Squibb Co., Seattle, WA (U.S. corp.)
APPL-NO: 08/219,518
DATE FILED: Mar. 29, 1994
ART-UNIT: 186
PRIM-EXMR: Donald E. Adams
LEGAL-REP: Merchant, Gould, Smith, Edell, Welter & Schmidt

US PAT NO: 5,580,756 [IMAGE AVAILABLE] L4: 1 of 13

ABSTRACT:
The invention identifies the B7 **antigen** as a ligand that is reactive with the CD28 receptor on T cells. Fragments and derivatives of the B7 **antigen** and CD28 receptor, including fusion proteins having amino acid sequences corresponding to the extracellular domains of B7 or CD28 joined to amino acid sequences encoding portions of human immunoglobulin C.gamma.1, are described. Methods are provided for using B7 **antigen**, its fragments and derivatives, and the CD28 receptor, its fragments and derivatives, as well as antibodies and other molecules reactive with B7 **antigen** and/or the CD28 receptor, to regulate CD28 positive T cell responses, and immune responses mediated by T cells. The invention also includes an assay method for detecting ligands reactive with cellular receptors mediating intercellular adhesion.

US PAT NO: 5,532,219 [IMAGE AVAILABLE] L4: 2 of 13
DATE ISSUED: Jul. 2, 1996
TITLE: Dapsone and promin for the treatment of dementia
INVENTOR: Patrick L. McGeer, Vancouver, Canada
Nobua Harada, Oku-gun, Japan
Horoshi Kimura, Otsu, Japan
Edith G. McGeer, Vancouver, Canada
Michael Schulzer, Vancouver, Canada
ASSIGNEE: The University of British Columbia, Vancouver, Canada (foreign corp.)
APPL-NO: 08/042,658
DATE FILED: Apr. 5, 1993
ART-UNIT: 125
PRIM-EXMR: Russell Travers

LEGAL-REP: Oyen Wiggs Green & Mutala

US PAT NO: 5,532,219 [IMAGE AVAILABLE] L4: 2 of 13

ABSTRACT:
This invention pertains to the novel use of 4,4'-diaminodiphenylsulfone and its didextrose sulfonate derivative and other closely related sulfones in the prevention and treatment of dementia (Alzheimer's disease). A method of preventing and treating dementia in a human being suffering from dementia which comprises administering to the human being a therapeutic amount of a substance selected from the group consisting of 4,4'-diaminodiphenylsulfone, its didextrose sulfonate derivative, and sulfoxone, sulfetrone and thiazolsulfone, and therapeutically acceptable salts thereof.

US PAT NO: 5,521,288 [IMAGE AVAILABLE] L4: 3 of 13
DATE ISSUED: May 28, 1996
TITLE: CD28IG fusion protein
INVENTOR: Peter S. Linsley, Seattle, WA
Jeffrey A. Ledbetter, Seattle, WA
Nitin K. Damle, Renton, WA
William Brady, Bothell, WA
ASSIGNEE: Bristol-Myers Squibb Company, Seattle, WA (U.S. corp.)
APPL-NO: 08/219,116
DATE FILED: Mar. 29, 1994
ART-UNIT: 186
PRIM-EXMR: Donald E. Adams
LEGAL-REP: Merchant, Gould, Smith, Edell, Welter & Schmidt

US PAT NO: 5,521,288 [IMAGE AVAILABLE] L4: 3 of 13

ABSTRACT:
The invention identifies the B7 **antigen** as a ligand that is reactive with the CD28 receptor on T cells. Fragments and derivatives of the B7 **antigen** and CD28 receptor, including fusion proteins having amino acid sequences corresponding to the extracellular domains of B7 or CD28 joined to amino acid sequences encoding portions of human immunoglobulin C.gamma.1, are described. Methods are provided for using B7 **antigen**, its fragments and derivatives, and the CD28 receptor, its fragments and derivatives, as well as antibodies and other molecules reactive with B7 **antigen** and/or the CD28 receptor, to regulate CD28 positive T cell responses, and immune responses mediated by T cells. The invention also includes an assay method for detecting ligands reactive with cellular receptors mediating intercellular adhesion.

US PAT NO: 5,474,771 [IMAGE AVAILABLE] L4: 4 of 13
DATE ISSUED: Dec. 12, 1995
TITLE: Murine monoclonal antibody (5c8) recognizes a human glycoprotein on the surface of T-lymphocytes, compositions containing same
INVENTOR: Seth Lederman, New York, NY
Leonard Chess, Scarsdale, NY
Michael J. Yellin, Riverdale, NY
ASSIGNEE: The Trustees of Columbia University in the City of New York, New York, NY (U.S. corp.)

APPL-NO: 07/792,728
DATE FILED: Nov. 15, 1991
ART-UNIT: 186
PRIM-EXMR: David L. Lacey
ASST-EXMR: Phillip Gambel
LEGAL-REP: John P. White

US PAT NO: 5,474,771 [IMAGE AVAILABLE] L4: 4 of 13

ABSTRACT:
This invention provides a monoclonal antibody which specifically recognizes and forms a complex with a protein located on the surface of activated T cells and thereby inhibits T cell activation of B cells. This invention also provides the monoclonal antibody 5c8 (ATCC Accession No. HB 10916).
This invention provides a human CD4.sup.- T cell leukemia cell line designated D1.1 (ATCC Accession No. CRL 10915) capable of constitutively providing contact-dependent helper function to B cells. This invention also provides an isolated protein from the surface of activated T cells, wherein the protein is necessary for T cell activation of B cells. This invention further provides an isolated, soluble protein from the surface of activated T cells, wherein the protein is necessary for T cell activation of B cells.

US PAT NO: 5,472,939 [IMAGE AVAILABLE] L4: 5 of 13
DATE ISSUED: Dec. 5, 1995
TITLE: Method of treating complement mediated disorders
INVENTOR: Douglas T. Fearon, Baltimore, MD
Lloyd B. Klickstein, Brookline, MA
Winnie W. Wong, Newton, MA
Gerald R. Carson, Wellesley, MA
Michael F. Concino, Newton, MA
Stephen H. Ip, Sudbury, MA
Savvas C. Makrides, Bedford, MA
Henry C. Marsh, Jr., Reading, MA
ASSIGNEE: The Johns Hopkins University, Baltimore, MD (U.S. corp.)
The Brigham and Women's Hospital, Boston, MA (U.S. corp.)
T Cell Sciences, Inc., Needham, MA (U.S. corp.)
APPL-NO: 08/138,825
DATE FILED: Oct. 19, 1993
ART-UNIT: 182
PRIM-EXMR: Garnette D. Draper
ASST-EXMR: John D. Ulm
LEGAL-REP: Pennie & Edmonds

US PAT NO: 5,472,939 [IMAGE AVAILABLE] L4: 5 of 13

ABSTRACT:
The present invention relates to the C3b/C4b receptor (CR1) gene and its encoded protein. The invention also relates to CR1 nucleic acid sequences and fragments thereof comprising 70 nucleotides and their encoded peptides or proteins comprising 24 amino acids. The invention further provides for the expression of the CR1 protein and fragments thereof. The genes and proteins of the invention have uses in diagnosis and therapy of disorders involving complement activity, and various immune system or inflammatory disorders. In specific embodiments of the present invention

detailed in the examples sections infra, the cloning, nucleotide sequence, and deduced amino acid sequence of a full-length CR1 cDNA and fragments thereof are described. The expression of the CR1 protein and fragments thereof is also described. Also described is the expression of a secreted CR1 molecule lacking a transmembrane region. The secreted CR1 molecule is shown to be useful in reducing damage caused by inflammation and in reducing myocardial infarct size and preventing reperfusion injury.

US PAT NO: 5,399,347 [IMAGE AVAILABLE] L4: 6 of 13
DATE ISSUED: Mar. 21, 1995
TITLE: Method of treating rheumatoid arthritis with type II collagen
INVENTOR: David E. Trentham, North Quincy, MA
Howard L. Weiner, Brookline, MA
David A. Hafler, West Newton, MA
ASSIGNEE: Autoimmune, Inc., Lexington, MA (U.S. corp.)
APPL-NO: 07/951,565
DATE FILED: Sep. 25, 1992
ART-UNIT: 183
PRIM-EXMR: Kay K. A. Kim
LEGAL-REP: Darby & Darby

US PAT NO: 5,399,347 [IMAGE AVAILABLE] L4: 6 of 13

ABSTRACT:
The present invention is directed to a method and pharmaceutical formulations for the treatment of **autoimmune** arthritis and animal models therefore in mammals, including humans, by the oral, enteral or by-inhalation administration of whole collagen protein or biologically active peptide fragments of collagen.

US PAT NO: 5,338,829 [IMAGE AVAILABLE] L4: 7 of 13
DATE ISSUED: Aug. 16, 1994
TITLE: Peptides derived from human immunodeficiency virus-1 GP160
INVENTOR: David B. Weiner, Penn Wynne, PA
Mark I. Greene, Penn Valley, PA
William V. Williams, Havertown, PA
ASSIGNEE: Trustees of the University of Pennsylvania, Philadelphia, PA (U.S. corp.)
APPL-NO: 07/932,078
DATE FILED: Oct. 19, 1992
ART-UNIT: 183
PRIM-EXMR: Christine M. Nucker
ASST-EXMR: Lynette F. Smith
LEGAL-REP: Woodcock Washburn Kurtz Mackiewicz & Norris

US PAT NO: 5,338,829 [IMAGE AVAILABLE] L4: 7 of 13

ABSTRACT:
This invention discloses novel polypeptides having an antigenic determinant or determinants immunologically cross-reactive with determinants of a glycoprotein having a molecular weight of approximately 41,000 daltons, and determinants of a glycoprotein having a molecular weight of approximately 160,000 daltons which are obtained from cells

infected with human immunodeficiency virus-1. The invention further discloses novel polypeptides having an antigenic determinant or determinants specific for a glycoprotein having a molecular weight of approximately 41,000 daltons obtained from cells infected with human immunodeficiency virus-1, the polypeptides further having an antigenic determinant or determinants immunologically cross-reactive with at least one glycoprotein having a molecular weight of 25,000 to 35,000 daltons, 45,000 daltons to 60,000 daltons, 80,000 to 100,000 daltons or 180,000 or 220,000 daltons, which are obtained from HSB, St, HeLa and human cells. The novel polypeptides of the invention are useful in methods of interfering with the effects of HIV-1 upon host cells having cell surface polypeptides capable of binding HIV-1. Methods of assay for HIV-1 infection are also disclosed. The invention also discloses peptides having amino acid sequences of about 10 to about 50 amino acids that correspond to at least a portion of an epitope of HIV and methods for developing such biologically active peptides.

US PAT NO: 5,304,474 [IMAGE AVAILABLE] L4: 8 of 13
DATE ISSUED: Apr. 19, 1994
TITLE: Hydrophobic peptide esters and amides
INVENTOR: Peter E. Lipsky, Dallas, TX
Dwain L. Thiele, Coppell, TX
ASSIGNEE: Board of Regents, The University of Texas System, Austin, TX (U.S. corp.)
APPL-NO: 07/684,010
DATE FILED: Apr. 11, 1991
ART-UNIT: 183
PRIM-EXMR: Ronald W. Griffin
ASST-EXMR: Pamela S. Webber
LEGAL-REP: Arnold, White & Durkee

US PAT NO: 5,304,474 [IMAGE AVAILABLE] L4: 8 of 13

ABSTRACT:
Esters or amides of a peptide, preferably a dipeptide, consisting essentially of natural or synthetic L-amino acids with hydrophobic side chains were found to have specific cellular toxicities. Preferable amino acids of the peptide are leucine, phenylalanine valine, isoleucine, alanine, proline, glycine or aspartic acid beta methyl ester. Preferable dipeptides are L leucyl L-leucine, L-leucyl L-phenylalanine, L-valyl L-phenylalanine, L-leucyl L-isoleucine, L-phenylalanyl L-phenylalanine, L-valyl L-leucine, L-leucyl L-alanine, L-valyl L-valine, L-phenylalanyl L leucine, L prolyl L-leucine, L-leucyl L-valine, L-phenylalanyl L-valine, L glycyl L-leucine, L-leucyl L-glycine, glycyl L-phenylalanine and L-aspartyl beta methyl ester L-phenylalanine. Most preferable dipeptides are L-leucyl L-leucine, L-leucyl L-phenylalanine, L-valyl L-phenylalanine, L-phenylalanyl L-leucine, L-leucyl L-isoleucine L-phenylalanyl L-phenylalanine and L-valyl L-leucine.

US PAT NO: 5,258,499 [IMAGE AVAILABLE] L4: 9 of 13
DATE ISSUED: Nov. 2, 1993
TITLE: Liposome targeting using receptor specific ligands
INVENTOR: Paula J. Konigsberg, Pasadena, CA
Leroy L. Richer, San Gabriel, CA
Paul G. Schmidt, San Marino, CA

Joseph A. Uliana, Sierra Madres, CA
ASSIGNEE: Vestar, Inc., San Dimas, CA (U.S. corp.)
APPL-NO: 07/850,019
DATE FILED: Mar. 10, 1992
ART-UNIT: 185
PRIM-EXMR: Jeffrey E. Russel
ASST-EXMR: Eric Linnell
LEGAL-REP: Adam Cochran, George A. Gilbert

US PAT NO: 5,258,499 [IMAGE AVAILABLE] L4: 9 of 13

ABSTRACT:
Delivery vehicle formulations comprise active agents encapsulated within liposomal vesicles to which are attached protein hormones (ligands) such as interleukin-2. The ligands are capable of showing affinity for specific cell receptors resulting in delivery of the encapsulated active agent to target cells, enabling delivery of active agents to particular cell populations in the treatment of conditions such as immune system disorders.

US PAT NO: 5,256,642 [IMAGE AVAILABLE] L4: 10 of 13
DATE ISSUED: Oct. 26, 1993
TITLE: Compositions of soluble complement receptor 1 (CR1) and a thrombolytic agent, and the methods of use thereof
INVENTOR: Douglas T. Fearon, Baltimore, MD
Lloyd B. Klickstein, Brookline, MA
Winnie W. Wong, Newton, MA
Gerald R. Carson, Wellesley, MA
Michael F. Concino, Newton, MA
Stephen H. Ip, Sudbury, MA
Savvas; C. Makrides, Bedford, MA
Henry C. Marsh, Jr., Reading, MA
ASSIGNEE: The Johns Hopkins University, Baltimore, MD (U.S. corp.)
Brigham and Women's Hospital, Boston, MA (U.S. corp.)
T Cell Sciences, Inc., Cambridge, MA (U.S. corp.)
APPL-NO: 07/588,128
DATE FILED: Sep. 24, 1990
ART-UNIT: 184
PRIM-EXMR: Robert A. Wax
ASST-EXMR: Stephen Walsh
LEGAL-REP: PenniPenni

US PAT NO: 5,256,642 [IMAGE AVAILABLE] L4: 10 of 13

ABSTRACT:
The present invention relates to compositions comprising soluble complement receptor 1 (CR1) and a thrombolytic agent. In a specific embodiment, the thrombolytic agent is anisoylated human plasminogen-streptokinase activator complex (ASPAC). The invention further relates to methods for treating thrombotic conditions in humans and animals by administering a composition comprising soluble CR1 and a thrombolytic agent. In particular, the compositions and methods are useful both for reducing reperfusion injury and ameliorating the other effects of myocardial infarction.

US PAT NO: 5,221,616 [IMAGE AVAILABLE] L4: 11 of 13
DATE ISSUED: Jun. 22, 1993
TITLE: Prevention of spontaneous complement activation in mammalian biological fluids
INVENTOR: William P. Kolb, Del Mar, CA
Linda M. Kolb, Del Mar, CA
John D. Tamerius, Carlsbad, CA
ASSIGNEE: Quidel Corporation, San Diego, CA (U.S. corp.)
APPL-NO: 07/219,922
DATE FILED: Jul. 15, 1988
ART-UNIT: 182
PRIM-EXMR: Sam Rosen
LEGAL-REP: Knobbe, Martens, Olson & Bear

US PAT NO: 5,221,616 [IMAGE AVAILABLE] L4: 11 of 13

ABSTRACT:
Selected polyhydroxyl compounds, comprising monosaccharides, disaccharides and oligosaccharides, as well as certain of their derivatives, are effective in preventing the spontaneous activation of complement in vitro. The effect is enhanced in the presence of an anticoagulant together with a divalent cation chelator. Addition of these compounds makes it possible to store clinical samples at conventional temperatures from -20.degree. to +22.degree. C. for extended periods prior to performing complement protein assays. Effective compounds are selected through a screening protocol which comprises the use of immunoassays for complement fragments together with an algorithm for computing effectiveness. Polyhydroxyl compounds extend similar protection from activation to coagulation proteins.

US PAT NO: 5,190,859 [IMAGE AVAILABLE] L4: 12 of 13
DATE ISSUED: Mar. 2, 1993
TITLE: Purification of LFA-3
INVENTOR: Michael Dustin, Boston, MA
Timothy Springer, Chestnut Hill, MA
ASSIGNEE: Dana-Farber Cancer Institute, Inc., Boston, MA (U.S. corp.)
APPL-NO: 07/421,292
DATE FILED: Oct. 3, 1989
ART-UNIT: 182
PRIM-EXMR: David Saunders
LEGAL-REP: Fish & Richardson

US PAT NO: 5,190,859 [IMAGE AVAILABLE] L4: 12 of 13

ABSTRACT:
A method of purifying LFA-3 using affinity chromatography. Purified LFA-3 is useful for quantitating or separating out CD-2-containing cells.

US PAT NO: 5,068,223 [IMAGE AVAILABLE] L4: 13 of 13
DATE ISSUED: Nov. 26, 1991
TITLE: Hydrophobic peptide esters and amides
INVENTOR: Peter E. Lipsky, Dallas, TX
Dwain L. Thiele, Dallas, TX
ASSIGNEE: Board of Regents, University of Texas System (U.S. corp.)

APPL-NO: 07/324,151
DATE FILED: Mar. 15, 1989
ART-UNIT: 181
PRIM-EXMR: Lester L. Lee
ASST-EXMR: Stephen B. Maebius
LEGAL-REP: Arnold, White & Durkee

US PAT NO: 5,068,223 [IMAGE AVAILABLE] L4: 13 of 13

ABSTRACT:
Esters or amides of a peptide, preferebly a dipeptide, consisting essentially of natural or synthetic L-amino acids with hydrophobic side chains were found to have specific cellular toxicities. Preferable amino acids of the peptide are leucine, phenylalanine valine, isoleucine, alanine, proline, glycine or aspartic acid beta methyl ester. Preferable dipeptides are L leucyl L-leucine, L-leucyl L-phenylalanine, L-valyl L-phenylalanine, L-Leucyl L-isoleucine, L-henylalanyl L-phenylalanine, L-valyl L-leucine, L-leucyl L-alanine, L-valyl L-valine, L-phenylalanyl L leucine, L prolyl L-leucine, L-leucyl L-valine, L-phenylalanyl L-valine, L glycyl L-leucine, L-leucyl L-glycine, glycyl L-phenylalanine and L-aspartyl beta methyl ester L-phenylalanine. Most preferable dipeptides are L-leucyl L-leucine, L-leucyl L-phenylalanine, L-valyl L-phenylalanine, L-phenylalanyl L-leucine, L-leucyl L-isoleucine L-phenylalanyl L-phenylalanine and L-valyl L-leucine.
The ester or amide of the dipeptide is most preferably alkyl, aralkyl or aryl a preferred alkylester is a methyl ester and may also be an ethyl ester or alkyl of up to about four carbon atoms such as propyl, isopropyl, butyl or isobutyl. Yet larger alkyl substituents may also be functional judging from the beta naphthyl substituent which is functional in certain embodiments.
These alkyl, aryl or arylkyl esters and amides of dipeptides consist essentially of amino acids with hydrophobic side chains may be used to deplete cytotoxic T-lymphocytes or natural killer cells from organisms, cell populations or tissues.

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